REMARKS

In the Office Action dated November 12, 2003, Claim 41 has been withdrawn from further consideration as allegedly drawn to a non-elected invention. Claims 2-6, 8-17, 25-26, 32, 38-40, and 42 are currently being examined.

The Examiner has maintained the "objection to reference to the drawings" in the specification. Claims 6 and 12-14 have been rejected under 35 U.S.C. §112, first paragraph, as allegedly lacking descriptive support. Claims 2, 4-5, 8, 10-11, 25-26, 32, 39-40 and 42 have been rejected under 35 U.S.C. §112, first paragraph, as allegedly lacking descriptive support. Claims 15-17, 25-26, 32, and 38 have been rejected under 35 U.S.C. §112, first paragraph, as allegedly lacking enabling support. The Examiner has objected to the amendment filed September 22, 2003 under 35 U.S.C. §132, as allegedly introducing new matter into the specification. Claims 2-6, 8-17, 25-26, 32, 38-40 and 42 have been rejected under 35 U.S.C. §101, as allegedly lacking a specific asserted utility and a substantial utility. Claims 2-5, 8-11, 25-26, 32, 39-40, and 42 have been rejected under 35 U.S.C. §101 as allegedly inoperable. Claims 2-6, 81-17, 25-26, 32, 38-40 and 42 have been rejected under 35 U.S.C. §112, first paragraph, as allegedly lacking usefulness and enablement. Claims 2, 4-5, 8, 10-11, 39, 40, and 42 have been rejected under 35 U.S.C. §112, first paragraph, as allegedly lacking descriptive support. Claims 2-5, 6, 8, 10-11, 12-14, 15-17, 32, 38-40 and 42 have been rejected under 35 U.S.C. 112, first paragraph, as allegedly lacking enabling support. Claims 25-26 have been rejected under 35 U.S.C. §112, first paragraph, as allegedly lacking enabling support. Claims 25 and 26 have been rejected under 35 U.S.C. §112, first paragraph, as allegedly lacking descriptive support.

The Examiner has maintained the objection to the references to the drawings in the specification. Specifically, the Examiner requires Applicant to delete all reference to the figures in the specification.

In an effort to favorably advance the prosecution of the present case, Applicant has deleted all mention of the figures in the specification, without prejudice. Accordingly, the objection to the references to the drawings in the specification is obviated and withdrawal thereof is respectfully requested.

Claims 6 and 12-14 have been rejected under 35 U.S.C. §112, first paragraph, as allegedly lacking descriptive support. Specifically, the Examiner contends that Applicant has not met the formal deposit requirements.

In response, Applicant has amended the specification to identify the deposits as mSAG, hSAG-mutant1, hSAG-mutant2 and hSAG clones, which bear ATCC accession numbers 98042, 98043, 98044 and 98045, respectively, and has had the same deposited at the ATCC (American Type Culture Collection, 10801 University Blvd., Manassas, VA 20110-2209).

Applicant further submits that all restrictions on availability of the above-mentioned deposited host cells to the public will be irrevocably removed upon the granting of the patent based upon the present application and the host cells will remain permanently available for a term of at least 5 years after the most recent request for the furnishing of a sample, and in any case, for a period of at least 30 years after the date of deposit or for the enforceable life of the U.S. patent whichever is longer. In the event that the host cells become non-viable or are inadvertently destroyed, such will be replaced with viable host cells of the same taxonomic description.

Accordingly, the rejection of Claims 6 and 12-14 under 35 U.S.C. § 112, first paragraph, is overcome, and withdrawal thereof is therefore respectfully requested.

Claims 2, 4-5, 8, 10-11, 25-26, 32, 39-40 and 42 have been rejected under 35 U.S.C. §112, first paragraph, as allegedly lacking descriptive support. Specifically, the Examiner contends that the claims are not drawn to a correlation of any structure and function as required. The Examiner also contends that it is unclear how the putative heme binding site could be involved with oxygen scavenging because Swaroop et al. (*Free Radical Biology and Medicine*, 1999, 27: 193-202) stated that "it appears that SAG protein does not bind heme." The Examiner further contends that the GCG program findings disclosed in the specification raise serious doubt as to the functions of the polypeptide encoded by the claimed polynucleotides, especially in view of the teachings of Swaroop et al.

In an effort to delineate the embodiments of the present invention more clearly and to expedite a favorable prosecution of the present application, Applicant has amended Claims 2, 8, 39-40 and 42. Support for the amendment can be found throughout the specification, on page 5, lines 9-15, for example.

Applicant respectfully submits that the claims as presently amended provide sufficient descriptive support as drawn to a correlation of structure and function of the claimed DNA molecule.

With respect to the Examiner's proposition that GCG program did not reveal any known functional domain of the polypeptide encoded by the claimed polynucleotides, Applicant respectfully submits that the GCG result alone does not negate the polypeptide's specific function discovered by the present invention. It is a common knowledge in the field of bioinformatics that if the GCG program did not reveal any known functional domain of the polypeptide encoded

by the claimed polynucleotides, there are two possibilities. First, since GCG is merely one database, it may not collect the functional domain disclosed in the present invention. For example, a functional domain may be revealed in the Pfam database but not in GCG. Second, even if no functional domain is revealed in all of the available databases, this result merely provides, at most, that the function of the protein is unknown before, which does not negate the function that is discovered by the present invention.

Applicant observes that the specification discloses novel isolated and purified DNA molecules of SAG genes, e.g., mouse SAG (SEQ ID NO: 1) and human SAG (SEQ ID NO: 3), and DNA molecules substantially similar to SEQ ID NO: 1 or SEQ ID NO: 3. See e.g., page 5, lines 9-15 of the instant specification. The specification also discloses that SAG proteins contain conserved zinc finger domain. See, e.g., page 15, line 28 to page 16, line 10 of the instant specificaiton. The specification further provides examples that demonstrate the function of the zinc finger domain in SAG protein. Specifically, the specification discloses that the zinc finger domain in the SAG protein protects cells against apoptosis. See, e.g., page 29, lines 24-30 of the instant specification. The specification also discloses that the heme binding site in the SAG protein acts as an oxygen radical scavenger to prevent oxygen radical induced damage. See, e.g., page 32, line 24 to page 33, line 3 of the instant specification.

Accordingly, Applicant submits that the present invention provides sufficient written description. Therefore, the rejection of Claims 2, 4-5, 8, 10-11, 25-26, 32, 39-40 and 42 under 35 U.S.C. §112, first paragraph, is overcome, and withdrawal thereof is respectfully requested.

Claims 2, 4-5, 20-22, 32, 38-40 and 42 have been rejected under 35 U.S.C. §112, first paragraph, as allegedly lacking enablement.

In the first instance, Applicant has added Claim 43, which is directed to a pharmaceutical composition comprising an expression vector that comprises the SAG protein disclosed in the present invention. Support of Claim 43 can be found throughout the present application, e.g., on page 12, line 33 to page 13, line 24. No new matter has been added.

Applicant respectfully submits that the present invention is directed to a novel gene and polypeptide derived therefrom encoding a redox-sensitive protein that protects cells from apoptosis and promotes cell growth. Applicant submits that the present invention is also directed to a pharmaceutical composition comprising an expression vector that comprises the SAG protein, which is operatively linked to a DNA molecule that promotes high level expression of the antisense strand of an isolated and purified DNA molecule in a tumor cell. Thus, Applicant submits that the expression of the antisense strand of the novel gene is useful for inhibition of tumor cell growth and therapeutic applications by gene therapy. A substantial and credible utility is clearly asserted in compliance with 35 U.S.C. § 101.

The Examiner alleges that Applicant has not disclosed a specific successful invention. Applicant assumes that such a rejection is intended to be premised on written description grounds. Applicant submits that the specification provides a sufficient teaching of successful identification and cloning of the SAG gene in both mice and humans (e.g., in Examples 1 and 2, on pages 13-16), successful expression and purification of the SAG proteins (e.g., in Example 6, on pages 18-20) and successful generation of single and double SAG mutants in heme binding sites and the zinc finger motif (e.g., in Example 8, on pages 21-23). The present application also provides a specific teaching that SAG expression protects cells from DNA fragmentation, a hallmark of apoptosis (e.g., in Example 16, on pages 29-30); that antisense SAG expression inhibits tumor cell growth (e.g., in Example 17, on pages 30-31); that

SAG can be used as a target in cancer gene therapy by expressing antisense SAG (e.g., in Example 18, pages 31-32); that SAG functions as an oxygen radical scavenger (e.g., in Example 19, on pages 32-33); that SAG mutations are cancer specific and can be used for diagnosing cancer (e.g., in Example 21, on pages 33-34); that SAG acts as a protector against lipid peroxidation (e.g., in Example 25, on pages 37-38); and that SAG protects against neuronal apoptosis (e.g., in Example 27, on pages 39-40). Therefore, the specification has provided sufficient description, ample guidance and detailed examples for those skilled in the art to make and use the claimed invention without an undue amount of experimentation.

Applicant respectfully submits that, as a matter of law, there is no requirement under 35 U.S.C. § 112, first paragraph, for the present application to include human or clinical trial data. The alleged unpredictability illustrated by the references cited by the Examiner, e.g., clinical efficacy in human gene therapy is, at best, general and cannot negate the specific, successful invention embodied in the present claims. Therefore, the cited references are irrelevant as to the utility, description or enablement of the claimed invention. Applicant further submits that 35 U.S.C. § 112 does not require an applicant to provide a working example for making and using the invention if the description of the invention itself is sufficient to permit one skilled in the art to make and use the invention, as is the case here.

Applicant acknowledges that additional experimentation, e.g., adjustment of certain parameters in different patients, may be required. However, such experimentation is routine to one skilled in the art and is not undue. Necessary experimentation is not determinative of the question of enablement; only *undue* experimentation is fatal under the provisions of 35 U.S.C. §112, first paragraph. *See, In re Wands*, 858 F.2d. 731, 736-737, 8 U.S.P.Q.2d 1400, 1404 (Fed. Cir. 1988).

Accordingly, Applicant submits that the present application embodies a substantial, credible utility and provides sufficient information for one skilled in the art to make and use the present invention, in the absence of undue experimentation. Therefore, the rejection of Claims 2-6, 8-17, 25-26, 32, 38-40, and 42 under 35 U.S.C. § 112, first paragraph, is overcome and withdrawal thereof is respectfully requested.

Claims 15-17, 25-26, 32, and 38 have been rejected under 35 U.S.C. §112, first paragraph, as allegedly lacking enabling support. Specifically, while recognizing that the specification provides DNA molecules that are substantially similar to those in SEQ ID NO:1 or SEQ ID NO:3, the Examiner contends that the specification does not teach how to use those "substantially similar" molecules. The Examiner contends that no real world use has been disclosed for proteins encoded by the claimed synthetic human SAG polynucleotides. The Examiner alleges that the use of antisense therapy as disclosed in the present application is unpredictable and thus not enabled. The Examiner contends that no SAG mutants associated with any primary cancer have been identified, although the Examiner acknowledges that SEQ ID NOS: 11 and 13 were detected in cancer cells lines. While recognizing that the specification discloses that there are deletion mutants in primary colon carcinoma samples, the Examiner contends that there is no teaching in the specification that the mutations were either the 7bp or 48bp deletions found in either SEQ ID NOS: 11 or 13. The Examiner also contends that cell culture data is not convincing. The Examiner further alleges that the specification would need more detail as how to make and use the invention, given the novelty of the claimed invention.

As submitted above, the present invention discloses how to use those molecules similar to those as set forth in SEQ ID NO: 1 or SEQ ID NO: 3. The present invention also discloses a pharmaceutical composition that employs proteins encoded by the claimed synthetic

human SAG polynucleotides. Thus, the specification has provided real world use and sufficient description, guidance and working examples for those skilled in the art to make and use the claimed invention. The rejection of Claims 15-17, 25-26, 32, and 38 under 35 U.S.C. §112, first paragraph, is overcome and withdrawal thereof is respectfully requested.

The amendment filed on September 22, 2003 has been objected to under 35 U.S.C. § 132, as allegedly introducing new matter into the specification. Specifically, the Examiner contends that the amendment of the specification on page 32 wherein the term "may" was deleted and the term "can" was substituted is not supported by the original disclosure. The Examiner requires that Applicant cancel the alleged new matter.

In response, Applicant resubmits that the specification on page 32 as disclosed in its original form asserts that oxidative buffering activity qualifies SAG as an oxygen radical scavenger. Such assertion, either by present tense, or using the term "may" or "can," sufficiently provides one in the skilled art with a predication that the protein will function as disclosed and claimed. Accordingly, the objection under 35 U.S.C. § 132 is overcome and withdrawal thereof is respectfully requested.

Claims 2-6, 8-17, 25-26, 32, 38-40 and 42 have been rejected under 35 U.S.C. §101, as allegedly lacking a specific asserted utility and a substantial utility.

The Examiner alleges that neither the specification nor any art of record teaches what the claimed polynucleotides or hybridization variants or encoded polypeptides are and what they do. The Examiner alleges that the specification does not teach a relationship to any specific diseases or establish any involvement in the etiology of any specific diseases.

As indicated above, Applicant respectfully submits that the present invention is supported by a substantial and well-established utility. The present invention is directed to a

novel gene and polypeptide derived therefrom encoding a redox-sensitive protein that protects cells from apoptosis and promotes cell growth. Applicant submits that the present invention is also directed to a pharmaceutical composition comprising an expression vector that comprises the SAG protein, which is operatively linked to a DNA molecule that promotes high level expression of the antisense strand of an isolated and purified DNA molecule in a tumor cell. The utility of using antisense DNA to treat tumors has been described in the art. Particularly, the specification, at page 2, lines 18-20, teaches that a better understanding of the molecular mechanisms of apoptotic induction will allow improved design of therapeutic drugs that either induce (anti-cancer) or inhibit (anti-aging) apoptosis. Such utilities are well-documented in the art and provide a credible and substantial utility for the instantly claimed invention.

Therefore, it is respectfully submitted that the specification has asserted at least one substantial utility or one well-established utility. As such, withdrawal of the rejection of Claims 2-6, 8-17, 25-26, 32 and 38-40 and 42 under 35 U.S.C. §101 is respectfully requested.

Claims 2-5, 8-11, 25-26, 32, 39-40, 42 have been rejected under 35 U.S.C. §101 as allegedly inoperable. The Examiner alleges that since the protein containing the putative heme binding site appears to not bind heme, the claimed invention is inoperative.

Applicant respectfully submits that Claims 2-5, 8-11, 25-26, 32, 39-40 and 42, as amended, and the newly added Claim 43 do not recite "heme binding site." Therefore, the rejection of Claims 2-5, 8-11, 25-26, 32, 39-40 and 42 under 35 U.S.C. §101 is overcome and withdrawal thereof is respectfully requested.

Claims 2-5, 8-11, 25-26, 32, 39-40, 42 have been rejected under 35 U.S.C. §101 as allegedly inoperable.

The Examiner contends that although the specification discloses that SEQ ID NO: 1 and 3 encode polypeptides that comprise putative heme binding sites and zinc ring finger domains, the complete complement of those coding regions would not encode said polypeptides since the nucleotide sequences would not be the same.

Applicant notes that the Examiner suggests that the claims be amended to recite, for example, "DNA molecules whose complete complement encodes polypeptide with heme and/or zinc finger domain" and that Hillier et al., (W38711)[1], "teaches a DNA molecule which comprises 100% identity to SEQ ID NO: 3, nucleotides 141-164 (which was found in us 09/509,779-3-copy-141-264.rst, result 5)" and that Marra et al., (AA230335)[2], "teaches a DNA molecule that comprises 99.5% of residues 63-634 of SEQ ID NO:1 (which was found in us 09/509,779-1.rst, result 50)."

In response, Applicant submits that DNA is a double-stranded molecule. A DNA molecule splits into two strands in the process of hybridization. A DNA molecule hybridizes to a protein coding sequence by the strand that is the complement to the protein coding regions. Thus, although the complement may not encode the protein since the nucleotide sequences would not be the same, the other strand of the DNA molecule can encode one or more domains on the protein. Thus, the present invention is fully operative. The rejection of Claims 2-5, 8-11, 25-26, 32, 39-40, 42 under 35 U.S.C. §101 is overcome and withdrawal thereof is respectfully requested.

Claims 2-6, 81-17, 25-26, 32, 38-40 and 42 have been rejected under 35 U.S.C. §112, first paragraph, as allegedly lacking utility and enablement. Specifically, the Examiner

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[1] Genbank Sequence Database (Accession W38711), National Center for Biotechnology Information, National Library of Medicine, Bethesda, Maryland, publicly available May 15, 1996.

[2] Genbank Sequence Database (Accession AA230335), National Center for Biotechnology Information, National Library of Medicine, Bethesda, Maryland, publicly available February 26, 1997.

alleges that since the claimed invention is not supported by a well established utility for the reasons set forth in the rejection under 35 U.S.C. §101 above, one skilled in the art clearly would not know how to use the claimed invention.

As submitted above, the claimed invention is supported by a credible and substantial utility or a well-established utility. Therefore one skilled in the art would know how to use the claimed invention. Thus, the rejection of Claims 2-6, 81-17, 25-26, 32 and 38-40 and 42 under 35 U.S.C. §112, first paragraph, is overcome. Withdrawal of the rejection is therefore respectfully requested.

Claims 2, 4-5, 8, 10-11, 39, 40, and 42 have been rejected under 35 U.S.C. §112, first paragraph, as allegedly lacking descriptive support. Specifically, the Examiner contends that the amendment submitted in the previous response that the claimed polypeptide "comprising at least one heme binding site and/or one zinc finger domain" has no descriptive support in the specification and the claims as originally filed.

Applicant submits that Claims 2, 4-5, 8, 10-11, 39, 40, and 42, as amended, do not recite heme binding site. Applicant also submits that there is sufficient description support for such recitation throughout the application, for example, at page 5, lines 14-15.

Therefore, the rejection of Claims 2, 4-5, 8, 10-11, 39, 40, 42 under 35 U.S.C. §112, first paragraph, is overcome and withdrawal thereof is respectfully requested.

Claims 2-5, 6, 8, 10-11, 12-14 (if not drawn to SEQ ID NO:3), 15-17, 32, 38-40 and 42 have been rejected under 35 U.S.C. 112, first paragraph, as allegedly lacking enabling support.

Specifically, the Examiner acknowledges that the specification enables a human SAG polynucleotide, SEQ ID NO: 3 and its complete complement. The Examiner, however,

contends that the specification does not provide enablement for any of the various polynucleotides associated with SEQ ID NO: 3 by hybridization conditions as claimed, for SEQ ID NO:1 or for any of the various polynucleotides associated with SEQ ID NO: 1 by hybridization conditions or for any of the synthetic deletion mutants claimed or for any of the deletion mutants of human cell lines claimed. The Examiner contends that there is no established nexus between the cell line mutants and any *in vivo* condition or disease.

Furthermore, the Examiner states that the claims as written do not require that the DNA molecule hybridize to the complete sequence of SEQ ID NOS:1 or 3, but only require that the polypeptide or protein encoded by said DNA molecule comprise at least one heme binding site and/or zinc finger domain. The Examiner also states that the minimum size for a stable complex is from 10 to 20 nucleotides. The Examiner concludes that given that the putative heme binding motif consists of 5 codons (or 18 nucleic acids) and the zinc finger domain consists of apparently 8 codons (or 24 nucleic acids), the claims allegedly read on fragments that do not possess either the structure or function contemplated for SEQ ID NOS: 1 and 3. The Examiner alleges that even if other segments of the molecules are left hanging, it would be expected that the claims as written would read on a whole universe of DNA molecules without the structure or function of SEQ ID NOS: 1 or 3.

In response, Applicant has amended the claims to recite that the polypeptide or protein maintains its function and possesses at least one zinc finger domain. The claims, as amended, recite sequences that are substantially similar to SEQ ID NOS: 1 or 3. Support for such amendment can be found throughout the specification, e.g., on page 5, lines 13-15.

Accordingly, the rejection of Claims 2-5, 6, 8, 10-11, 12-14, 15-17, 32, 38-40 and 42 under 35 U.S.C. 112, first paragraph, is overcome and withdrawal thereof is respectfully requested.

Claims 25-26 have been rejected under 35 U.S.C. §112, first paragraph, as allegedly lacking enabling support. Specifically, the Examiner acknowledges that the specification enables a diagnostic method for identifying colon cancer cells by detecting 7bp and/or 48bp deletion mutation of SEQ ID NO: 3 with PCR assay using primers that flank the region amplified by SEQ ID NOS 7 and 8. The Examiner contends, however, that the specification does not provide enablement for a method for identifying any cancer cells by detecting mutations in a gene encoding a redox-sensitive protein that protects cells from apoptosis comprising subjecting an extract of the cells to PCR amplification using primers that have a sequence comprising the DNA sequences of Claims 2, 3, 8, 9, or 15.

Applicant submits that Claims 25-26 are directed to diagnostic assay for identifying cancer cells by detecting cells containing mutations in a gene encoding a redox-sensitive protein that protect cells from apoptosis. The Examiner seems to require data from a human cancer diagnosis using SAG as a marker. Applicant submits that human data is simply not required for enabling support under 35 U.S.C. § 112, first paragraph.

Notably, the specification provides a sufficient teaching of the successful identification and cloning of the SAG gene in both mice and humans (e.g., in Examples 1 and 2, on pages 13-16), successful expression and purification of the SAG proteins (e.g., in Example 6, on pages 18-20) and successful generating single and double SAG mutants in heme binding sites as well as the zinc finger motif (e.g., in Example 8, on pages 21-23). The present application also provides specific teachings that SAG expression protects cells from DNA fragmentation, a

hallmark of apoptosis (e.g., in Example 16, on pages 29-30); that antisense SAG expression inhibits tumor cell growth (e.g., in Example 17, on pages 30-31); that SAG can be used as a target in cancer gene therapy by expressing antisense SAG (e.g., in Example 18, pages 31-32); that SAG functions as an oxygen radical scavenger (e.g., in Example 19, on pages 32-33); that SAG mutations are cancer specific and can be used for diagnosing cancer (e.g., in Example 21, on pages 33-34); that SAG acts as a protector against lipid peroxidation (e.g., in Example 25, on pages 37-38); and that SAG protects against neuronal apoptosis (e.g., in Example 27, on pages 39-40). Therefore, the present invention provides sufficient description, guidance and working examples for one skilled in the art to make the DNA molecules and mutants thereof use them in detecting cancer cells.

Applicant respectfully submits that, as a matter of law, there is no requirement under 35 U.S.C. § 112, first paragraph, for the present application necessarily to include human or clinical trial data. The alleged unpredictability illustrated by the references cited by the Examiner, e.g., clinical efficacy in human gene therapy is, at best, general and cannot negate the specific, successful invention embodied in the present claims. Therefore, the cited references are irrelevant as to the enablement of the claimed invention. 35 U.S.C. § 112 does not even require an applicant to provide a working example for making and using the invention if the description of the invention itself is sufficient to permit one skilled in the art to make and use the invention, as is the case here.

Therefore the rejection of Claims 25-26 under 35 U.S.C. §112, first paragraph, is overcome and withdrawal thereof is respectfully requested.

Claims 25 and 26 have been rejected under 35 U.S.C. §112, first paragraph, as allegedly lacking descriptive support. Specifically, the Examiner states that Claims 25-26 are

drawn to a diagnostic assay for identifying cancer cells by detecting cells containing mutations in a gene encoding a redox-sensitive protein that protects cells from apoptosis, comprising subjecting said nucleic acid molecules to amplification with primers and determining whether the resulting PCR product contains a mutation.

The Examiner alleges that the specification does not describe genes encoding a redox-sensitive protein that protects cells from apoptosis and that are useful for identifying cancer cells and "mutations" that are diagnostic for identifying cancer cells required to practice the method of Claims 25-26 in a manner that satisfies the standards in either *University of California v. Eli Lilly and Co.*, 119 F.3d 1559, 43 USPQ2d 1398 (Fed. Cir. 1997) or *Enzo Biochem, Inc. v. Gen-Probe Inc.*, 296 F3d 1316, 63 USPQ2d 1609 (Fed. Cir. 2002).

In response, Applicant respectfully submits that the present invention is directed to a novel gene and polypeptide derived therefrom encoding a redox-sensitive protein that protects cells from apoptosis and promotes cell growth. Applicant submits, as admitted by the Examiner, that the Federal Circuit in *Enzo* has clarified that a DNA molecule can be adequately described without disclosing its complete structure. The *Enzo* Court has adopted the standard that the written description requirement can be met by showing sufficiently detailed, relevant identifying characteristics. *Enzo*, 296 F3d at 1324, 63 USPQ2d at 1613. The *Enzo* Court has held that examples of such relevant characteristics include complete or partial structure, other physical and/or chemical properties, functional characteristics when coupled with a known or disclosed correlation between function and structure, or some combination of such characteristics. *Id.*

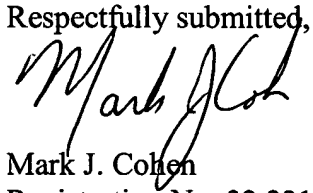
Applicant reiterates that the specification discloses identification and cloning of the SAG gene in both mice and humans (e.g., in Examples 1 and 2, on pages 13-16), expression

and purification of the SAG proteins (e.g., in Example 6, on pages 18-20) and generation of single and double SAG mutants in heme binding sites and the zinc finger motif (e.g., in Example 8, on pages 21-23). The present application also provides specific teachings that SAG expression protects cells from DNA fragmentation, a hallmark of apoptosis (e.g., in Example 16, on pages 29-30); that antisense SAG expression inhibits tumor cell growth (e.g., in Example 17, on pages 30-31); that SAG can be used as a target in cancer gene therapy by expressing antisense SAG (e.g., in Example 18, pages 31-32); that SAG functions as an oxygen radical scavenger (e.g., in Example 19, on pages 32-33); that SAG mutations are cancer specific and can be used for diagnosing cancer (e.g., in Example 21, on pages 33-34); that SAG acts as a protector against lipid peroxidation (e.g., in Example 25, on pages 37-38); and that SAG protects against neuronal apoptosis (e.g., in Example 27, on pages 39-40). Therefore, the specification has provided sufficiently detailed, relevant identifying characteristics of the claimed DNA molecules.

Accordingly, the rejection of Claims 25 and 26 under 35 U.S.C. §112, first paragraph, is overcome, and withdrawal thereof is respectfully requested.

In view of the foregoing amendments and remarks, it is firmly believed that the subject application is in condition for allowance, which action is earnestly solicited.

Respectfully submitted,



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